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HIV: Its Development Within the Human Population

Jeffrey S. Charno

Beginning in the late 1970's, physicians in New York and San Francisco reported the increasing occurrence of a rare type of cancer (Kaposi's sarcoma), and a variety of infections including pneumocystis pneumonia among previously healthy young homosexual males (Brandt, 1993:547). The physicians were baffled by these mysterious deaths because these illnesses are usually controlled by the body's defenses (Shubert, 1992:1). Due to the unusual character of these diseases, epidemiologists began to search for the characteristics that might link the cases (Brandt, 1993:547). This research eventually led to the description of Acquired Immunodeficiency Syndrome (AIDS) in 1981. Evidence indicates that the causal virus had been spreading undetected in numerous populations during the 1970's (Brandt, 1993:547). Early studies showed that homosexual men, recipients of blood transfusions or blood products (often haemophiliacs), and intravenous drug users were at greatest risk for acquiring the disease (Brandt, 1993:547). For this reason, researchers focused on a common infectious agent that could be transmitted sexually or through blood (Brandt, 1993:547). In 1983, Frenchman Luc Montagnier and American Robert Gallo independently identified a previously undescribed human retrovirus (Brandt, 1993:547). This new virus was named Human Immunodeficiency Virus (HIV).

Tests to detect antiviral antibodies, specific for HIV were first devised in 1984 (Brandt, 1993:547). These tests do not detect the virus directly, instead they measure the high levels of antibodies which are produced upon infection (Brandt, 1993:547).

This paper is about HIV, the virus which is transmitted between humans, and may manifest into AIDS. It will discuss the biology and life cycle of HIV, the evolution of HIV, transmission of HIV, how AIDS can affect human population dynamics, and current research on the treatment of AIDS.

BIOLOGY AND LIFE CYCLE OF HIV

Like all viruses, HIV is an intracellular parasite. The virus particle is inactive and therefore cannot reproduce or do any damage until it enters a host cell (Weber and Weiss, 1988:75). HIV is an RNA (ribonucleic acid) human retrovirus, which means that it reverses the normal flow of genetic information by using an enzyme, reverse transcriptase (Dalgleish, 1990:122). In cells, the genetic material is DNA (deoxyribonucleic acid). When genes are expressed, the DNA is first transcribed into messenger RNA (mRNA), which then serves as a template for translation to proteins (Haseltine and Wong–Staal, 1988:14). The viral genetic information, now as double–stranded DNA, migrates to the cell nucleus, where a third viral enzyme, called an integrase, splices the HIV genetic information into the host cell's DNA (Haseltine and Wong–Staal, 1988:14). Once established in the individual, the incorporated viral genome is a permanent component of the hosts' genome. The second half of the viral life cycle, which involves the production of new virus particles, takes place randomly and may be delayed for up to 10 years. Only certain infected cells are triggered to produce mature viral particles. The symptoms of this part of the life cycle is what is known as "full blown AIDS" (Haseltine and Wong–Staal, 1988:14).

When individuals are first infected with HIV they generate a strong immune response to the virus (Weber and Weiss, 1988:84). They produce antibodies to all the viral proteins and their immune system activates the various types of killer and scavenger cells that are part of any normal immune response (Weber and Weiss, 1988:84). However, once infection has occurred these immune responses do not stop the progress of the disease, due to the lack of active T4 cells which would normally prevent infection. Thus the HIV afflicted individual is now susceptible to attack by any other pathogen (viral,
bacterial, or fungal) and unlike a healthy individual, is unable
to defend him or herself.

**PATTERNS OF HIV TRANSMISSION**

HIV is transmitted only through the direct exchange of
blood, semen or vaginal secretions (Shubert, 1992:7). To date
four routes of transmission have been identified: (1)
unprotected sexual contact, (2) unscreened transfusions or
infusions of blood or blood products, (3) sharing of
intravenous needles, (4) congenital or perinatal transmission
from a woman to her fetus or newborn (Shubert, 1992:7).
Infected blood poses a threat only if it comes in contact with
an open wound or when there is an inoculation (Shubert,

Brandt (1993:547) has identified three epidemiological
patterns of HIV transmission, which follow geographic
boundaries. Pattern I includes North America, Western Europe,
Australia, New Zealand and many urban centres in Latin
America. In these highly developed areas, transmission has
been predominantly among homosexual and bisexual males.
Since the introduction of widespread blood screening,
transmission via blood now occurs principally among
intravenous drug users who share syringes. Although there is
no evidence of widespread infection among the heterosexual
populations in these countries, heterosexual transmission of
the virus from those infected via intravenous drug use has
increased.

In pattern II countries, which consist of sub-Saharan
Africa and Latin America, transmission of HIV occurs through
heterosexual contact. In some urban areas, up to 25 percent of
sexually active adults are infected. Transfusion is a dominant
form of transmission because universal screening of blood is
not routine in these countries.

Pattern III countries include North Africa, the Middle East,
Eastern Europe, Asia, and the Pacific. In these locales, HIV
was not present until the mid 1980's. Therefore, infection has
been the result of contact with infected individuals from pattern
I and II countries, or importation of infected blood.

In the early to mid 1980's HIV transmission to
haemophiliacs was first noted (Anderson and Gazzard,
1990:91). This mode of transmission has since been reduced,
but not eliminated, by the heating of plasma to 56°C, and the
The number of individuals affected in Pattern I, II, and III
countries has been steadily increasing. It has been suggested
that by the year 2000 the number of HIV infected individuals
will be between 40 and 100 million (Wain-Hobson,

**THE EVOLUTION OF HIV**

HIV, which causes AIDS in humans, is an example of
"fast-forward" evolution (Myers and Korber, 1994:211).
Because of their high error rate during replication, RNA
viruses show high host diversity and a rate of evolution about
a million times faster than that of DNA organisms (Penny,

Researchers in 1984, while examining the origins of HIV,
began to look for similar viruses in non–human primates
(Essex and Kanki, 1988:27). At this same time, veterinary
pathologists in primate research centres reported symptoms
similar to AIDS in captive Asian macaques (Essex and Kanki,
1988:30). HIV related antibodies in the macaques were isolated
and the causal virus was named Simian Immunodeficiency
Virus (SIV) (Essex and Kanki, 1988:30). There is now strong
evidence suggesting that the HIVs and SIVs have evolved from
an unknown precursor within the highly diverse family of
retroviruses (Myers and Korber, 1994:212). The world–wide
prevalence of these retroviruses suggests that they are old, but
their exact age and time of divergence has yet to be determined

Like humans infected with HIV, Asian macaques infected
with SIV suffer a decrease in T4 cells, and the animals die of
infections very similar to those seen in human AIDS patients
(Essex and Kanki, 1988:30). Further studies revealed that
Asian macaques in the wild were not infected with SIV, unlike
those in captivity (Essex and Kanki, 1988:30). The research
then focused on Africa for a possible link in African primates.
Blood samples from chimpanzees, baboons, and African green
monkeys were analyzed for antibodies that reacted to the SIV
virus from Asian macaques (Essex and Kanki, 1988:30). The
results found no evidence of SIV infection in the chimpanzees
or baboons. However, over 50 percent of the wild African
green monkeys studied showed evidence of an SIV infection,
but exhibited no symptoms of AIDS (Essex and Kanki,
1988:30).

The fact that SIV appeared harmless in African green
monkeys but lethal to captive Asian macaques, indicated that
some SIV strains might have evolved toward co–existence
with their monkey hosts (Essex and Kanki, 1988:32). Most
retroviruses tend to co–exist with their host species in a way
that allows both to survive, and thus have become non–
pathogenic (Essex and Kanki, 1988:32). It appears that HIV
and SIV may be most pathogenic when they first afflict a new
species, thus selecting for the survival of the virus and the
host strains (Essex and Kanki, 1988:32).

The primate research done in the early 1980's led to the
discovery of a new retrovirus which was infecting people in
West Africa (Essex and Kanki, 1988:34). This new West
African retrovirus was somewhat similar to the HIV virus in
Central Africa, Europe, and the U.S. However, it was found to
be more closely related to SIV (Anderson and Gazzard,
1990:88). This new discovery led to the original AIDS virus
being named HIV–1, and the West African virus being named
HIV–2 (Essex and Kanki, 1988:34). The fact that HIV–2 is
more similar to SIV than it is to HIV–1, indicates that primate
and human viruses share evolutionary roots and that there may
have been inter–species infection (Essex and Kanki, 1988:34).

Attempts to reconstruct the evolutionary tree for HIV has
been difficult for two reasons: (1) the high variation in
nucleotide sequence among HIV isolates caused by rapid
mutations, (2) there is only a sparse "fossil record" for HIV
and limited historical and epidemiological data (Smith et al.,
1988:573). Despite this, Smith et al. (1988) compared the
envelope gene sequences from both old and new HIV isolates
and calculated that HIV–1 and HIV–2 split off from a common
ancestor as recently as 40 years ago. However Sharp and Li
(1988:315) dispute the validity of the 40 year split. Rather,
they used the conserved DNA sequence of the polymerase gene
to estimate that the divergence of HIV–1, from HIV–2, and
SIV occurred between 140 and 160 years ago. Furthermore,
because HIV–1 and HIV–2 seem to have evolved at similar
rates, Sharp and Li (1988) estimated that HIV–2 and SIV

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diverged only about 30 years ago. They argue that since all three viruses are from a common ancestor, all three were pathogenic. Because HIV–1 and HIV–2 cause AIDS whereas SIV does not, SIV probably lost its pathogenicity after the divergence. Conversely, if the three viruses were originally non–pathogenic, then HIV–1 and HIV–2 must have acquired their pathogenicity independently after divergence (Sharp and Li, 1988:315). This would be less likely. The similarity of one virus to the others and its likely phylogenetic origin can also be determined by comparing the genetic coding for those parts of the viruses which are essential for its survival, and therefore in which mutation rates would be low (Anderson and Gazzard, 1990:88). Using this methodology Anderson and Gazzard (1990) found that HIV–2 may have emerged from a simian group of viruses quite recently, but there is no corresponding recent ancestor for HIV–1. This may imply that HIV–1 has been present in the community for much longer than earlier research had suggested (Anderson and Gazzard, 1990:88).

**MUTATIONS AS A FORM OF EVOLUTION IN HIV**

HIV replication has three steps at which mutations are likely to occur: (1) the viral DNA polymerase lacks the error–correcting features found in analogous cellular enzymes, (2) the copying errors it makes in converting viral RNA into a single DNA strand and then synthesizing the complementary strand are not corrected, (3) the cellular RNA polymerase that makes the genetic material for new virions also does not correct its own errors (Haseltine and Wong–Staal, 1988:24). These three steps are common to all retroviruses, and they have been found to produce an average of about one mutation per replication cycle (Haseltine and Wong–Staal, 1988:24).

Although point mutations are the most frequent source of genetic diversity in most organisms, retroviruses are capable of generating a whole series of additional complex genetic changes that are of potential importance (Wain–Hobson, 1994:199). These other changes include duplications, deletions, substitutions with insertion, and deletions with insertions (Wain–Hobson, 1994:199). In other organisms, the errors are often checked and corrected, however this is not the case in retroviruses. Retroviruses accumulate huge numbers of variants, and HIV studies reflect the first analyses of retrovirus evolution (Wain–Hobson, 1994:204). Also, given the large number of individuals infected, it is only a matter of time before new pathogenic variants of HIV emerge (Wain–Hobson, 1994:204). At this high error rate, new HIV variants can also develop during the infection of one individual (Haseltine and Wong–Staal, 1988:24). These mutations may affect the progress of the disease in an individual since any change in the virus may alter its ability to cause disease. Changes in an HIV protein may also enable the virus to evade the immune response directed at the protein, and thus the new variant of HIV would be favoured by natural selection (Haseltine and Wong–Staal, 1988:24).

**EFFECTS OF HIV ON THE HUMAN POPULATION**

The AIDS epidemic is likely to have a substantial impact on the world demographic pattern. The disease primarily affects young and middle–aged adults between the ages of 20 to 49 (Brandt, 1993:550). These individuals are the reproductively and economically active members of society. If a substantial number of them exhibit a decrease in biological fitness, the population structure could be affected in two ways. First, a decrease in the total number of individuals of this generation will affect the older generation. In the population age pyramid of a society, the older cohorts depend on the middle–aged cohorts for support (Smelser, 1988:345). This support maximizes the number of individuals that have long life expectancies. By decreasing the number of young and middle–aged individuals, fewer old–aged individuals will have long life spans (Brinkerhoff and White, 1988:514).

Second, the decrease in fitness of young to middle–aged adults can result in a decrease in the general fertility rate of a population (Smelser, 1988:347). Thus, if fewer individuals produce offspring, a second change in population dynamics is evident, namely a decrease in the number of young cohorts (Smelser, 1988:347). The overall effect would be a decrease in world total population (Brinkerhoff and White, 1988:513).

Since the concentration of HIV positive individuals is unequal in different regions of the world, the impact of the disease would be apparent as a change in the population mosaic. In addition, some genetic alleles are more prevalent in certain regions of the world. By decreasing the number of individuals with these alleles, these traits could, in effect, be removed from the gene pool thus changing both the genotype and phenotype frequencies observed in future generations. This would have a direct impact on the evolution of Homo sapiens.

**TREATMENT OF AIDS**

In 1984, when HIV was shown to be the cause of AIDS, many researchers doubted that a cure would be found. The reason is that HIV can hide in the cells of the central nervous system, where it is protected by the blood–brain barrier, which many drugs can not penetrate (Brandt, 1993:548). Even if a drug could penetrate the barrier, brain cells already damaged by the virus may never be repaired (Yarchoan et al., 1988:85).

At present, researchers are experimenting with several different drugs to stop the virus from spreading in an infected individual. To date, the only drug which is showing promising results is azidothymidine (AZT).

Any therapeutic agent used against a pathogen, must either kill the pathogen or stop it from multiplying, without harming the infected host (Yarchoan et al., 1988:85). Often the drug used will attack a biochemical pathway unique to the pathogen (Yarchoan et al., 1988:85). Viruses, however, present a more difficult problem because they are simply packets of genetic material (RNA in HIV) protected by glycoproteins and lipids (Yarchoan et al., 1988:87). An RNA virus cannot replicate on its own. Rather, it must rely on another organisms' genetic machinery and biochemical pathways in order to replicate (Yarchoan et al., 1988:87).

AZT is being used to treat severely infected AIDS patients. AZT is not a cure for AIDS, rather it prolongs the patients' life. AZT was originally synthesized in 1964 by Jerome P. Horwitz of the Michigan Cancer Foundation as a potential anti-cancer drug, which failed (Yarchoan et al., 1988:92). However, when doctors used AZT against AIDS, it inhibited the production of viral DNA by two mechanisms: competitive inhibition and chain termination (Yarchoan et al., 1988:92). In competitive inhibition, AZT triphosphate binds to reverse transcriptase at a site that ordinarily binds to physiological nucleoside triphosphates (Yarchoan et al.,
1988:92). In chain termination, reverse transcriptase is deceived into incorporating AZT triphosphate in a growing chain of viral DNA in place of normal thymidine triphosphate (Yarchoan et al., 1988:92). When the viral DNA chain tries to add the next link, it is prevented because AZT triphosphate lacks the hydroxyl (OH) group that is needed to copy the chemical bond to the next link (Yarchoan et al., 1988:92).

AZT was first tested on an AIDS patient on July 3, 1985, and it was shown to increase the survival time and improve the quality of life for the patient (Yarchoan et al., 1988:87). After taking AZT for several weeks, the patient gained weight and had an increased number of helper T4 cells (Yarchoan et al., 1988:96). AZT can reduce the amount of HIV present in patients, but these improvements are only temporary and, because of side effects that occur in some patients, some researchers doubt whether the benefits outweigh the side effects (Yarchoan et al., 1988:96). The main problem with AZT is its toxicity, especially to bone marrow, such that patients on AZT often develop anaemia (a decrease in red blood cells) (Yarchoan et al., 1988:97). Despite the side effects, AZT has been shown to increase the survival time of patients with advanced AIDS by about one year (Yarchoan et al., 1988:96).

In March of 1987, the U.S. Food and Drug Administration approved AZT as a prescription drug for severe HIV infected patients (Shubert, 1992:8). However, AZT represents only the beginning to finding a cure for AIDS, and it is hoped that with further research a vaccine will be developed.

**SUMMARY**

In the early 1980's young homosexual males were dying of a mysterious illnesses, which should have been fought by their own immune systems. Research into the causes of these deaths led to the discovery of a new disease, named AIDS. Early studies showed that the disease was passed through the blood or by sexual intercourse. In 1983 the virus which causes AIDS was named HIV.

HIV is an RNA retrovirus, which contains an enzyme (reverse transcriptase) to reverse its genomic information (RNA) to DNA, the type of genomic information found in its host. The virus invades by attaching to the host cell and injects its core. Enzymes change the viral RNA into DNA, and then incorporate it into the hosts' genome. Once an individual is infected, the number of functioning T4 cells decreases and thus hampers the immune systems' ability to fight off infections. The virus can remain dormant in the host for up to 10 years, and then spontaneously manifest itself as "full blown" AIDS.

Current research has proposed three possible evolutionary patterns for HIV all of which include its relationship to SIV. All three theories suggest that the viruses HIV-1, HIV-2, and SIV share a common ancestor, but the authors of these theories disagree on the time of divergence and the order of events. All three use different methods for determining the phylogeny, thus further research is required to definitively support one theory over another.

HIV has the potential to affect world population dynamics. Since the disease is most prevalent among the reproductively fertile members of society, it can affect that group directly, as well cohorts both in younger and older groups. Also, since the percentage of individuals afflicted varies with location, the effect of the disease will be to change the population mosaic of each society differently.

Researchers are currently studying various drugs and their affects on HIV in the hopes of finding a cure or vaccine. To date, the only drug which seems to be effective is AZT. AZT inhibits the spread of HIV by halting the growth of the viral DNA chain. Once AZT is incorporated into the chain there is nothing the virus can do to correct it.

AZT is not a cure for AIDS, it only prolongs the individuals life by about one year. It is hoped that through future research an AIDS vaccine will be developed, which will bring an end to this devastating disease.

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